properties at high blood concentrations. MacLeod and Lamberts[3] have shown the dopamine agonist effects of high concentrations of haloperidol and pimozide on prolactin release. Seeman[4] has shown that very high concentrations of some antipsychotic drugs may enhance presynaptic release of neurotransmitters, including dopamine.

Alternatively, at high concentrations of antipsychotic drugs maximal dopaminergic blockade may occur. Additional dosage increments would not produce further dopamine blockade, but other pharmacological effects, such as anticholinergic activity, might continue to increase and might be responsible for the presentations described. Toxic psychoses have been associated with high doses of anticholinergic drugs,[5] usually combined with moderate doses of antipsychotic drugs. The episodes reported in these studies also appear to have been characterised by excitement and aggression, as in our cases.

Requests for reprints to Dr Bridges.

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Pseudomonas fluorescens cross-infection due to contaminated humidifier water

Ventilated patients in intensive therapy units are particularly susceptible to respiratory infections by Gram-negative organisms. In the case of *Pseudomonas aeruginosa* this susceptibility has been associated with the administration of broad-spectrum antibiotics, pre-existing immunosuppression, and corticosteroids.[1]

Cross-infection has often been by way of contaminated ventilator equipment. This may be due to inadequate disinfection of the apparatus after use.[2] Dilution of topical anaesthetics with deionised tap water has produced an outbreak of *Ps cepacia* respiratory tract infection.[3] *Pseudomonas* species are of a low order of pathogenicity and are rarely implicated as a cause of clinical infection. Nevertheless, they have been known to cause a granulomatous pneumonitis under certain circumstances.[4]

Patients and methods

During routine twice-weekly bacteriological monitoring of an intensive therapy unit *Ps fluorescens* was consistently isolated from the tracheal aspirates of those patients who had been ventilated for four days or more. Twenty-nine consecutive admissions for mechanical ventilation were finally analysed. Only seven of these patients were ventilated for four days or more, and from all these *Ps fluorescens* was isolated. *Ps fluorescens* was identified by means of both the analytical profile index (API Products Ltd) and fluorescein-pyocyanin production. All isolates had the same analytical profile index number, produced fluorescein only on King's medium, and had the same antibiogram.

On more detailed bacteriological examination of the intensive therapy units, *Ps fluorescens* was isolated from the humidifier water of all the ventilators in use, from the inspiratory tubing, and from the pre-filter expiratory tubing. At this time it was also noted that the temperature of the humidifiers, which are connected between the ventilator and the patient, was between 38°C and 41°C. Bacteriological examination was then performed on the humidifiers, humidifier water, and ventilator tubing before use. Only the distilled water, used to fill the humidifiers, yielded *Ps fluorescens* (20 organisms/l).

The distilled water was delivered from the pharmacy in 8-litre plastic containers; one container was used for all the patients. The organism was isolated from the empty containers but not from freshly prepared distilled water collected into sterile specimen bottles directly from the distillation unit in the pharmacy. *Ps fluorescens* was not isolated on environmental screening of the unit. Suspicion was then directed to the 8-litre plastic containers. These were not sterilised or dried out before refilling and were therefore presumed to be acting as a reservoir for the *Ps fluorescens*.

In the light of these findings commercially prepared bottles of sterile water are now used. Each patient is strictly allocated his own bottle of sterile water for "topping up" the humidifier. Furthermore, the humidifiers are now operated at the manufacturer's recommended temperature of 50°C to maintain the inspired gases at the patient end at 37°C (Cape Engineering Limited, personal communication). The humidifiers and tubing are also renewed daily. After instituting this practice only two unrelated isolates of *Ps fluorescens* were found among the next 104 patients admitted to the unit.

Comment

Ps fluorescens causes clinical infection infrequently, but when this occurs treatment with appropriate antibiotics may be ineffective. Of the seven patients who had been ventilated for four days or more only one, a chronic bronchitic, developed a clinical infection which required treatment with gentamicin. The other six patients had supportive treatment with active and frequent physiotherapy and were gradually weaned from their ventilators. This cautionary tale shows how a change in clinical practice may lead to a potentially dangerous contamination of ventilators. It illustrates too the advantages of continuous bacteriological monitoring and of a close link between clinician and bacteriologist, both in prevention and eradication of cross-infection.

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Low bioavailability as a cause of apparent failure of dihydroergotamine in orthostatic hypotension

Dihydroergotamine (DHE) has been used to improve postural hypotension in patients with autonomic insufficiency. It constricts capacitance vessels and so lessens venous pooling on standing.[1] Comparative pharmacokinetic investigations with tritium-labelled dihydroergotamine after oral and intravenous administration to man suggest that on average about 30% of an oral dose is absorbed.[2] We describe two patients whose postural hypotension was greatly improved with intravenous DHE but whose apparent failure to respond to DHE by mouth was due to low bioavailability.

Case reports

(1) A 56-year-old diabetic man had symptoms of severe postural hypotension (standing systolic blood pressure less than 70 mm Hg) despite good diabetic control, a high sodium diet, and fludrocortisone 0·9 mg/day. Thirty minutes after being given DHE 10 μg/kg intravenously his standing systolic blood pressure had risen by 109 mm to 169 mm Hg and the postural drop in pressure had lessened from 84 to 17 mm Hg. Postural symptoms were relieved for several hours. Single doses of up to 10 mg DHE by mouth failed to reproduce the results of intravenous treatment nor was